60% of cases, and culture, which is positive in about [illegible]

Cause	*Opening pressure*	*Protein*	*Glucose*	*Cell counts (WBCs)*	*Other findings*
Normal CSF	50–180 mmHg	15–45 mg/dL	40–85 mg/dL	0–5	
Bacterial meningitis	Elevated	(Marked increase) >250 mg/dL	Normal to marked decrease.	100–60,000 Predominance of neutrophils	Positive CSF culture and Gram stain. Blood positive cultures in 50%.
Viral meningitis	Usually normal	<100 mg/dL (moderate increase)	Normal	<100 Predominance of lymphocytes	Culture is negative; PCR for certain agents like HSV, JC virus. Elevated RBCs in

CamScanner

TB meningitis	Variable	(Moderate to marked increase) 50–1,000 mg/dL	<40 mg/dL (Low)	10–500 Predominance of neutrophils early and lymphocytes late	Positive for AFB and MTB DNA.
Fungal meningitis	Variable	(Moderate to marked increase) 25–500 mg/dL	<40 mg/dL (Low)	25–500 Predominance of lymphocytes	Positive culture India ink positive
Guillain–Barré syndrome	Normal	100–1,000 mg/dL	Normal	Normal	This is called albuminocytologic dissociation

CamScanner

Some important points in neurology:

Six points should be considered in a patient who presented with symptoms and signs of neurological impairment;

- ✓ What is the lesion?
- ✓ Where is the lesion?
- ✓ What is the cause of lesion?
- ✓ What functional impairment occurs due to the lesion?
- ✓ How to confirm the lesion?
- ✓ What are the different treatment options?

What is the lesion?

This is usually obvious from history and examination, like hemiparesis/plegia, paraparesis/plegia, quadriparesis/plegia, monoparesis or plegia, distal and proximal weakness and weakness in the restricted distribution.

Where is the lesion?

Localization of the lesion is very important step in neurology. Localization needs broad knowledge of neuroanatomy but some important points are as follow;

- First step is to determine whether it is upper motor neuron lesion or lower motor neuron lesion. Some of the important signs that distinguish UMN from LMN lesions are tabulated:

Sign	*UMN lesion*	*LMN lesion*
Atrophy	None	Severe
Fasciculations	None	Common
Tone	Spastic	Decreased
Weakness distribution	Pyramidal/ Regional	Distal/ Segmental
Reflexes	Hyperactive	Absent
Babinski sign	Present	Absent

- In UMN lesion, distal muscle groups are affected more than the proximal ones and axial movements are spared until the lesion is severe and bilateral. Weakness from UMN lesion occurs particularly in the extensors and abductors of the upper limb and the flexor of the lower limb.
- LMN lesions occur due to the disorders of cranial nerve nuclei or anterior horn of the spinal cord or from dysfunction of the axons of these neurons. LMN weakness depends on whether involvement is at the level of the anterior horn cells, nerve root, limb plexus or peripheral nerve. Only muscles supplied by the affected structures are weak.
- At the level of lesion, there is always lower motor neuron lesion and below the level of lesion, there is an UMN lesion.

CamScanner

- LMN lesion signs are present on the side of lesion (ipsilateral), whereas UMN lesion can be on the same side or on the opposite side depending whether the lesion is below or above the pyramid, respectively. If UMN lesion is below the pyramid, ipsilateral UMN signs will be present below the level of lesion and if the UMN lesion is above the pyramid, contralateral UMN signs will be present below the level of lesion.
- A hemiparesis results from an UMN lesion above the midcervical spinal cord. Presence of other signs will help to localize the lesion:
  - Language disorder=> cortical lesion
  - Pure motor dense hemiparesis => Internal capsule/ cerebral peduncle
  - Uncrossed hemiplegia => If there is UMN facial weakness and limb weakness present on same side, it is called uncrossed hemiplegia and the level of lesion is always above the brain stem level and most probably in internal capsule
  - Crossed hemiplegia => if LMN facial weakness is present on one side and UMN weakness of limbs occur on the opposite side, it is called crossed hemiplegia and the level of lesion is in the pons. Any ipsilateral cranial nerve signs and contralateral hemiplegia is called crossed hemiplegia and the lesion is present in brain stem e.g. Weber syndrome in which there is ipsilateral 3rd nerve palsy with contralateral hemiplegia. It occurs when lesion is in midbrain.
  - Paraparesis is mostly caused by an intraspinal lesion. Diseases of the cerebral hemispheres that produce acute paraparesis are anterior cerebral artery infarction, cortical venous thrombosis and hydrocephalus.
  - Level of lesion in spinal cord is assessed by sensory level.
  - Acute distal weakness suggests LMN or peripheral nerve disease.
  - Myopathy often produces symmetric weakness of the pelvic or shoulder girdle muscles.
  - Neuromuscular junction disorders cause fatigable weakness.

What is the cause of lesion?

The time course over which neurologic symptoms arise and evolve provides crucial information about the causes of the lesion and differential diagnosis. Some of the important pathophysiological causes are as below:

- Vascular=> sudden in onset
- Infectious=> slowly progressive
- Neoplastic=> progressive worsening of symptoms
- Toxic and metabolic
- Immune mediated=> relapsing remitting course

Functional impairment due to lesion:

The impact of lesion on daily activities, bowel and bladder functions, loss of occupation, language problem and vision problems.

Confirmation of lesion;

The typical findings of some of the important CNS lesions should be memorized like CSF findings, MRI findings of MS, NCS findings of GBS etc.

Treatment of lesion:

Important treatment steps like thrombolysis for acute infarction, plasmapheresis or IVIG for GBS etc. should be memorized.

CamScanner

Laboratory confirmation requires serologic tests to detect specific antibodies to B burgdorferi in serum by ELISA.

Treatment of Lyme disease

Manifestations	*Treatment*
Tick bite	Observe only
Erythema migrans	Doxycycline 100 mg BD for 10-14 days or Amoxicillin 500 mg TDS for 2-3 weeks or Cefuroxime axetil 500 mg BD for 2-3 weeks
Facial palsy without meningitis	As for erythema migrans
Facial palsy with meningitis	Ceftriaxone 2g IV OD or Penicillin G, 18-24 million units daily in 6 divided doses or Cefotaxime 2g TDS **All for 2-4 weeks**
Cardiac disease	IV treatment as for meningitis
Arthritis	Oral doxycycline for 4 weeks, if response, continue for further 4 weeks. If no response to oral doxycycline, IV ceftriaxone for 4 weeks.

CamScanner

choice for this is triclabendazole. Bithionol is second line drug.

Treatment for helminthics:

Organism/Disease	*Drugofchoice*	*Alternatetherapies*
Schistosomiasis	Praziquantel 1 or 2 doses	Oxamniquine
Fasciolahepatica	Triclabendazole	Bithionol, Nitazoxanide
Clonorchis sinensis	Praziquantel	Albendazole, tribendimidine
Paragonimus westermani	Praziquantel	Bithionol, triclabendazole
Taenia saginata, Taenia solium, diphyllobothrium latum, Hymenolepis nana	Praziquantel	Niclosamide
Cysticercosis	Albendazole	Praziquantel
Echinococcosis	Albendazole	Surgical resection

CamScanner

palsy and

Some important brainstem stroke syndromes;

Syndrome	Signs Symptoms	Localization	Artery involved
Weber	Ipsilateral 3rd nerve+ contralateral hemiparesis including the lower face	Medial midbrain/ Cerebral Peduncle	Deep penetrating branch of PCA
Benedikt	Ipsilateral 3rd nerve + contralateral involuntary movements like intention tremors, hemi chorea, and hemiathetosis.	Ventral midbrain involving red nucleus	Paramedian Penetrating branches of basilar artery or deep penetrating branch of PCA
Nothnagel	Ipsilateral 3rd nerve palsy + contralateral dysmetria and limb ataxia	Superior cerebellar peduncle	Deep penetrating branch of PCA
Foville	Ipsilateral 6th and 7th nerve palsy(lateral gaze palsy+ facial weakness) with or without contralateral hemiplegia	Caudal pontine tegmentum involving facial colliculus	Pontine perforator branches of basilar artery
One-and-a-half	Ipsilateral 6th nerve palsy+ bilateral internuclear opthalmoplegia	Paramedian pons involving medial longitudinal fasciculus	Paramedian perforator off the basilar artery
Dejerine	Ipsilateral tongue weakness with contralateral hemiparesis with or without contralateral loss of proprioception and vibration	Medial medulla	Vertebral artery or anterior spinal artery

CamScanner

Hyperlipo-proteinemia	Synonyms	Defect	Increased lipoprotein	Main symptoms	Treatment	
Type I	Familial hyperchylomicronemia	Decreased lipoprotein lipase	Chylomicrons	Acute pancreatitis, lipemia retinalis, eruptive skin xanthomas, hepatosplenomegaly	Diet control	
Type IIa	Familial hypercholesterolemia	LDL receptor deficiency	LDL	Xanthelasma, arcus senilis, tendon xanthomas	Bile acid sequestrants, statins, niacin	
Type IIb	Familial hyperlipidemia	Decreased LDL receptor and increased ApoB	LDL and VLDL		Statins, niacin, fibrate	
Type III	Broad beta disease or dysbetalipoproteinemia	Defect in Apo E 2 synthesis	IDL	Tubercruptive xanthomas palmar xanthomas	Fibrate, statins	
Type IV	Familial hypertriglyceridemia		Increased VLDL production and decreased elimination	VLDL	Can cause pancreatitis at high triglyceride levels	Fibrate, niacin, statins

Type V	Mixed hyperlipoproteinemia	Increased VLDL production and decreased LPL	VLDL and chylomicrons		Niacin, fibrate

CamScanner

Features	Epileptic seizure	Pseudoseizure
Trigger	Defined reflex mechanism like sleep withdrawal	Emotional
Prodrome	Stereotypical, short	Variable, Prolonged
Eyes	Initially eyes open	Eyes closed tightly, Bell phenomena
Falls	Abrupt	Protective movements with no injury
Pupils	Mydriasis without light reflex	Mydriasis can occurs
Automatisms	Repetitive, oroalimentary	Undulating, changing, crying, head shaking
Convulsions	Tonic clonic and decrescendo	Crescendo or waxing and waning
Injury	Injury present, burns	Auto aggressive injuries
Tongue biting	Lateral tongue, inner cheeks	Tip of tongue only
Enuresis	Possible	Rare
Breathing	Apnea, postictal snoring	Normal, sometimes breath holding with normal oxygenation
Facial color	Cyanotic	Normal
Duration	<2 minutes	>5 minutes
Amnesia	Complete	Remember the episode
Postictal	Slow orientation, Todd paralysis, Babinski sign	Astonishing awakening, crying, screaming

CamScanner

Occurrence	Can occurs anytime	Only present when under observation
Serum prolactin	Raised	Normal

Ascariasis	Albendazole/mebendazole	Pyrantal pamoate, ivermectin
Trichuris trichiura	Albendazole or mebendazole	Ivermectin
Hook worms	Albendazole or mebendazole	Pyrantal pamoate, levamisole
Strongyloidiasis	Ivermectin	Albendazole, thiabendazole
Enterobius vermicularis	Albendazole or mebendazole	Pyrantal pamoate
Trichinosis	Albendazole or mebendazole	
Toxocariasis	Albendazole	Ivermectin
Cutaneous larva migrans	Albendazole	Ivermectin
Wuchereria bancrofti	Diethylcarbamazine	Albendazole, ivermectin
Onchocerciasis	Ivermectin	Doxycycline
Loiasis	Diethylcarbamazine	Albendazole

CamScanner

Can be used	*Use if CD4> 200*	*Contraindicated*
Hep A	MMR	Cholera
Hep B	Varicella	Oral polio
HiB	Yellow Fever	Intranasal Influenza
Influenza parenteral		BCG
Japanese Encephalitis		
Meningococcus		
Pneumococcus		
Polio parenteral		
Td		
Rabies		

diarrhea but exacerbates fatty

Difference between bile acid diarrhea and fatty acid diarrhea:

Features	*Bile acid diarrhea*	*Fatty acid diarrhea*
Extent of Ileal disease	Limited	Extensive
Ileal bile acid absorption	Reduced	Reduced
Fecal bile acid excretion	Increased	Increased
Bile acid synthesis compensated by hepatic synthesis	Yes	No
Bile acid pool size	Normal	Reduced
Intra-duodenal bile acid	Normal	Reduced
Steatorrhea	None or mild	>20g
Response to cholestyramine	Yes	No
Response to low fat diet	No	Yes

CamScanner

Bleeding disorders with lab findings:

Platelets	*Bleedingtime*	*PT*	*APTT*	*Causes*
Normal	Normal	Normal	Normal	Vasculitis, telangiectasia, scurvy,
Low	Prolonged	Normal	Normal	Thrombocytopenia due to any cause
Normal	Prolonged	Normal	Normal	Glanzmann thrombasthenia, platelets storage pool disease, antiplatelets therapy, uremia, cirrhosis.
Normal	Prolonged	Normal	Prolonged	Von Willebrand disease
Normal	Normal	Prolonged	Normal	Vitamin K deficiency, cirrhosis, warfarin therapy
Normal	Normal	Normal	Prolonged	Hemophilia A and B, APLS,
Low	Prolonged	Prolonged	Prolonged	DIC

CamScanner

A.dominant hypocalcemia	Normal	Normal	Normal	Normal
Hypomagnesaemia	Normal	Normal	Low	Normal

CamScanner

Explanation: The most common extraarticular manifestation is keratoconjunctivitis Sicca. Pleurisy is th most common respiratory manifestation and pericarditis is the most common cardiac manifestation Scleritis is the most serious eye complication of RA. Neuropathy is a rare complication.

Organ	*Mostcommon*	*Otherpresentation*
Cardiac	Atherosclerosis	Myocarditis, pericarditis, MR, AR
Pulmonary	Pleurisy	Exudative pleural effusion, ILD, pulmonary nodules, bronchiolitis obliterans, Caplan syndrome
Eyes	Sicca syndrome (overall most common)	Episcleritis, scleritis, keratitis, uveitis
Renal	Amyloidosis induced NS	
Nerve	Cervical myelopathy	Mononeuritis multiplex, carpal tunnel syndrome
Skin	Rheumatoid nodules	

CamScanner

Blood	Anemia of chronic disease	Felty syndrome, thrombocytosis
Other	Vasculitis, bacterial overgrowth	

CamScanner

High

102) A 54 year old female presented with joint pain and intermittent purpuric rash for 8 months. She also complains of intermittent abdominal pain, malaise, fever, and weight loss. Examination showed a purpuric rash involving her calves and thighs. Labs were done which showed urea 56, creatinine 1.9, ESR 70 and negative RA factor and ANA. Urinalysis showed protein +, RBCs ++. C-ANCA and P-ANCA are positive. What is the most likely diagnosis?

a) Mixed cryglobulinemia
b) Wagner granulomatosis
c) Microscopic polyangitis
d) Churgg Strauss syndrome
e) Systemic lupus erythematosus

Ans: Microscopic polyangitis

Explanation: Wegener's granulomatosis (WG) and microscopic polyangiitis (MPA) are closely related systemic vasculitis. Both are associated with ANCA and are grouped as ANCA-associated vasculitis. ANCA directed against proteinase 3 (PR3) are more common in WG and those directed against myeloperoxidase (MPO) are associated with MPA. Involvement of the ears, nose, and throat such as granulomatous rhinitis is a common finding in WG and is observed in only a minority of patients with MPA. A sparing of upper respiratory tract is a differentiating feature of MPA from WG. Treatment is same as for WG with good response to steroids and immunosuppression than WG.

CamScanner

Antibodies associated with neurological disorders:

Antibodies	*Disorder*
Ganglioside M1 (GM1) antibody.	multi-focal motor neuropathy
IgG anti-GQ1b ganglioside antibodies	Miller-Fisher syndrome Bickerstaff's brainstem encephalitis Acute ophthalmoparesis Ataxic Guillain-Barre syndrome
Ganglioside asialo GM1 (anti-GA1) antibodies.	Sensorimotor neuropathies.
anti-Hu antibodies	Paraneoplastic neurologic syndromes associated with small-cell carcinoma of the lung.
voltage-gated potassium channel antibodies	Limbic encephalitis
voltage-gated calcium channel antibodies	Lambert-Eaton myasthenic syndrome
Anti-Yo antibodies	Paraneoplastic cerebellar degeneration associated with breast cancer or tumors of the ovary, endometrium, and fallopian tube.
glutamic acid decarboxylase (GAD) antibodies	Stiff-person syndrome
AChE receptor antibodies and anti-MuSK antibodies	Myasthenia Gravis
Aquaporin-4 Autoantibodies	Neuromyelitis optica (NMO)
GM1, GD1a IgG antibodies	Acute motor axonal neuropathy
Anti-Ri	Paraneoplastic cerebellar degeneration, brainstem encephalitis, opsoclonus-myoclonus
Sulfatide IgM (monoclonal or polycional)	Sensory neuropathy

CamScanner

Step 1: Check the pH. Is there alkalemia or acidemia present?

If pH < 7.35 => *acidemia*

If pH > 7.45 => *alkalemia*

This is usually a primary disorder, *pH can be normal with mixed opposite disorders* for which you need PCO2, HCO3 and anion gap.

Step 2: Is the disturbance respiratory or metabolic? What is the relationship between the direction change in the pH and the direction of change in the PaCO2?

- In primary respiratory disorders, the pH and PaCO2 change in opposite directions;
- In metabolic disorders the pH and PaCO2 change in the same direction.

Acidosis	Respiratory	pH low	CO2 high	High HCO3 compensation in
Acidosis	Metabolic	pH low	HCO3 low	Low PCO2 compensation in
Alkalosis	Respiratory	pH high	Co2 low	Low HCO3 compensation in
Alkalosis	Metabolic	pH high	HCO3 high	High PCO2 compensation in

Step 3. Is there appropriate compensation for the primary disturbance? *Usually compensation does not return the pH to normal.*

Disorder	Expected compensation
Metabolic acidosis	PCO2 = 1.5*ΔHCO3 + 8
Acute respiratory acidosis	Increase in HCO3= ΔPCO2/10
Chronic respiratory acidosis	Increase in HCO3= 3.5(ΔPCO2/10)
Metabolic alkalosis	Increase in PCO2= 40+0.6(ΔHCO3)
Acute respiratory alkalosis	Decrease in HCO3= 2(ΔPCO2/10)
Chronic respiratory alkalosis	Decrease in HCO3 = 5(ΔPCO2/10)

If the observed compensation is not the expected compensation, it is likely that more than one acid/base disorder is present.

- **Step 4.** Calculate the anion gap (if a metabolic acidosis exists): AG= Na-(Cl- + HCO3-)
- Normal anion gap Is approximately 12 meq/L.

CamScanner

hypoparathyroidism.

Causes of hypocalcemia:

Disease	PO4	Alk Phos	PTH	Vit D
Vitamin D deficiency	Low	High	High	Low
Hypoparathyroidism	High	High	Low	Normal
Renal disease	High	High	High	Low
Pseudohypoparathyiodism	High	Normal	High	Normal
Vitamin D resistance	Low	Normal	Low	Normal

CamScanner

JVP waveform abnormalities:

Waveforms	Causes
Cannon (irregular, large) a waves	Complete heart block
Absent a waves	Atrial fibrillation
Dominant v waves	Tricuspid regurgitation
Prominent x descent	Acute cardiac tamponade Constrictive pericarditis
Absent x descent	Atrial fibrillation
Sharp y descent	Severe tricuspid regurgitation Constrictive pericarditis
Slow y descent	Tricuspid stenosis Right atrial myxoma

19)

Characteristic	*Arterial*	*Venous*	*Neurogenic*
Quality of pain	Cramping	Bursting	Electric shock like
Onset	Gradual, consistent	Gradual and rarely acute	Can be immediate but inconsistent
Relieved by	Standing still	Elevation of leg	Sitting and bending forward
Location	Muscle groups (buttocks, thigh, leg)	Whole leg	Poorly localized, but mostly above knee
Time to relief	< 5 mins	Variable	Upto 30 mins

CamScanner

HIV Stage	Neurological Complications
Early (CD4 >500)	HIV meningitis Acute inflammatory demyelinating syndrome
Middle (CD4 200–500)	HIV-associated dementia Mononeuritis multiplex HIV-associated myopathy or neuropathy
Late (CD4 <200)	CNS toxoplasmosis Progressive multifocal leukoencephalopathy Primary CNS lymphoma Cryptococcal meningitis HIV vacuolar myelopathy CMV ventriculitis and VZV vasculitis

CamScanner

Total anterior circulation stroke (TACS):

A total anterior circulation stroke (TACS) is a large cortical stroke affecting the areas of the brain supplied by both the middle and anterior cerebral arteries. All three of the following need to be present for a diagnosis of a TACS:

- Unilateral weakness (and/or sensory deficit) of the face, arm and leg
- Homonymous hemianopia
- Higher cerebral dysfunction (dysphasia, visuospatial disorder).

Partial anterior circulation stroke (PACS):

A partial anterior circulation stroke (PACS) is a less severe form of TACS, in which only part of the anterior circulation has been compromised. Two of the following need to be present for a diagnosis of a PACS:

- Unilateral weakness (and/or sensory deficit) of the face, arm and leg
- Homonymous hemianopia
- Higher cerebral dysfunction (dysphasia, visuospatial disorder

Posterior circulation syndrome (POCS):

A posterior circulation syndrome (POCS) involves damage to the area of the brain supplied by the posterior circulation (e.g. cerebellum and brainstem). One of the following need to be present for a diagnosis of a POCS:

- Cranial nerve palsy and a contralateral motor/sensory deficit
- Bilateral motor/sensory deficit
- Conjugate eye movement disorder (e.g. horizontal gaze palsy) •Cerebellar dysfunction (e.g. vertigo, nystagmus, ataxia)
- Isolated homonymous hemianopia.

Lacunar stroke (LACS):

A lacunar stroke (LACS) is a subcortical stroke that occurs secondary to small vessel disease. There is no loss of higher cerebral functions (e.g. dysphasia). One of the following needs to be present for a diagnosis of a LACS:

- Pure sensory stroke
- Pure motor stroke
- Sensory-motor stroke
- Ataxic hemiparesis

Brainstem stroke syndromes are already described above.

CamScanner

- In patients with hypoalbuminemia, the normal anion gap is lower than 12 meq/L; the "normal" anion gap in patients with hypoalbuminemia is about 2.5 meq/L lower for each 1 gm/dL decrease in the plasma albumin concentration.
- If the anion gap is elevated, consider calculating the osmolal gap in compatible clinical situations. It is used to narrow down the differentials of high anion gap metabolic acidosis. OSM gap = measured OSM – (2[Na+] +glucose/18 + BUN/2.8. The osmolal gap should be less than 10 in common causes like DKA, Uremia and lactic acidosis. High osmolal gap is present in ethanol, methanol, ethylene glycol and isopropyl alcohol poisoning.

Step 5: If an increased anion gap is present, assess the relationship between the increase in the anion gap and the decrease in HCO3. Assess the ratio of the change in the anion gap (ΔAG) to the change in HCO3 (ΔHCO3): ΔAG/ΔHCO3. It is called ***delta ratio or delta delta.***

$$\text{DELTA RATIO} = \frac{\Delta \text{ anion gap}}{\Delta\ HCO_3^-} = \frac{[AG - 12]}{[24 - HCO_3^-]}$$

< 0.4	Hyperchloraemic non-anion gap metabolic acidosis
0.4 - 0.8	HAGMA + NAGMA [may be isolated renal failure]
1 -2	uncomplicated HAGMA
> 2	Metabolic acidosis w/ pre-existing elevated HCO3- [metabolic alkalosis or respiratory acidosis]

- This ratio should be between 1.0 and 2.0 if an uncomplicated anion gap metabolic acidosis is present.
- If this ratio falls outside of this range, then another metabolic disorder is present. If ΔAG/ΔHCO3 < 1.0, then a concurrent non-anion gap metabolic acidosis is likely to be present. If ΔAG/ΔHCO3> 2.0, then a concurrent metabolic alkalosis is likely to be present.
- Another important equation is **delta gap (DG),** which is calculated as ΔAG-ΔHCO3-.
- If delta gap > 6 => additional metabolic alkalosis
- If delta gap – 6 – 6 => no additional metabolic disorder
- If delta gap <-6 => additional non-anion gap metabolic acidosis

Mixed and complex acid base disorders:

R. Acidosis+ M. acidosis	Low pH, low HCO3, high PCO2	Cardiac arrest, intoxication, multiorgan failure
R. Alkalosis+ M. Alkalosis	High pH, high HCO3, low PCO2	Cirrhosis with diuretics, vomiting in pregnancy, over ventilation in COPD
R. Acidosis + M. Alkalosis	pH normal, PCO2 high, HCO3 high	COPD with diuretics, vomiting or NG aspiration, severe hypokalemia
R. Alkalosis + M. Acidosis	pH normal, HCO3 low, PCO2 high	Salicylate poisoning, renal failure with pneumonia, sepsis.
M. Acidosis+m.alkalosis	pH normal, HCO3 normal, PaCO2 normal, increase delta gap.	Uremia or DKA with vomiting or diuretics.

CamScanner